ARTERIAL COMPLIANCE IS DECREASED IN PATIENTS WITH CONGESTIVE HEART FAILURE

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Systemic vascular resistence is elevated in patients with congestive heart failure (CHF), however, it is not known whether compliance of conduit arteries is abnormal in this condition. Accordingly, we measured arterial compliance in 18 normal volunteers (NL) and 10 age-matched patients with CHF (40±2 vs 44±4 yrs;p=NS). Carotid artery diameter (d), assessed directly by computerized high resolution B-mode ultrasonography, and systolic blood pressure (p) were measured in each subject. The relative change in diameter (Δd) and systolic blood pressure (Δp) during an isometric handgrip maneuver allowed us to calculate the arterial compliant properties in each group. We determined strain ($\Delta d/d_1$), distensibility ($2\Delta d/d_1/\Delta p$), compliance ($\Delta d/d_1/2\Delta p\,d_1 2$), and stiffness ($\log p_2/p_1/\Delta d/d_1$). Measurements were obtained at an equivalent level of tension ($p_1 \cdot d_1/2$) in each group.

	NL	CHF	p
Tension (mmHg.mm)	510±20	450±37	N.S.
Strain (index)	0.08±0.01	0.03±0.01	<0.01
Distensibility (10^{-3}/kPa)	60.2±6.7	28.8±4.2	<0.01
Compliance ($10^{-7}m^2$/kPa)	17.9±2.2	9.3±1.6	=0.01
Stiffness (index)	16.3±1.7	43.4±5.2	<0.01

Conclusion: In patients with congestive heart failure, arterial compliance and distensibility are decreased and stiffness is increased. These abnormalities may adversely affect left ventricular systolic function in this disorder.

LOW PREVALENCE OF HYPOMAGNESEMIA IN CHF AS A RESULT OF ACE INHIBITION.

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Previous reports of the prevalence of hypomagnesemia in Congestive Heart Failure (CHF) range from 7 to 37%. However, these studies were performed prior to the widespread use of angiotensin converting enzyme inhibitors (ACE) in CHF. We therefore evaluated 188 patients (pts) referred for the treatment of CHF since 1988, and detected hypomagnesemia (Mg <1.6 mg/dl) in only 11 pts (6%). In order to determine whether this low prevalence was related to ACE inhibition, we compared patients taking ACE (ACE+) and not receiving ACE (ACE-) regarding mean (±SEM) Mg, ejection fraction by nuclear ventriculography (EF,%), New York Heart Association functional class (NYHA), dose of furosemide (Fur,mg/day), creatinine (Cr, mg/dl), and blood urea nitrogen (BUN, mg/dl) where *=p<0.05.

	Mg	EF	NYHA	Fur	BUN	Cr
ACE+	2.01±.03	21±1	2.83±.07	113±8	31±2	1.9±.3
ACE-	1.90±.04*	26±2*	2.54±.09*	77±11*	24±3	1.4±.1

Hypomagnesemia was found in 6/55 (11%) ACE- pts, a prevalence similar to previous reports. However, only 5/133 (4%) ACE+ pts had Mg < 1.6. There were no differences between the groups regarding age, sex, or serum sodium concentration. Despite higher diuretic doses (Fur) and evidence of more advanced failure (by EF and NYHA), ACE+ pts had higher Mg, suggesting that ACE use decreases Mg excretion. However, the tendency toward better renal function in ACE- pts (consistent with milder CHF) may also have contributed to increased Mg excretion.

In conclusion, we detected hypomagnesemia in only 6% of CHF pts, a prevalence less than previously reported. Since ACE+ pts have higher Mg despite more advanced CHF, the decreased prevalence of hypomagnesemia is probably due to the widespread use of ACE inhibitors in CHF.

Wednesday, March 6, 1991
Poster Displayed: 2:00PM-5:00PM
Author Present: 4:00PM-5:00PM
Hall F, West Concourse
Consequences of Ischemia

REGIONAL WALL MOTION IN PATIENTS WITH UNSTABLE ANGINA PECTORIS IN A PLACEBO CONTROLLED THROMBOLYSIS STUDY.

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To assess the possible benefit of thrombolysis (T) in unstable angina, we estimated the ischemia related regional wall motion (RWM) in a placebo controlled trial studying the effect of APSAC. 126 Pts with the typical history of unstable angina and typical ECG changes were included. Pts with previous myocardial infarction were excluded. Angiography I was performed within 3 hours after randomization. Thereafter, study medication was given. Angiography II was repeated after 12-24 hours.
The RWM was semi-quantitatively evaluated. A numerical score was assigned to each vessel related segment according to the degree of asynergy, varying from 1 for normal to 6 for dyskinesis. For both the LAD and RCX/RCA the score of each segment was used to calculate a total WM score (TWMS) by adding the scores of 4 segments. The coronary diameter stenosis (CDS) was quantitatively calculated. Results of both treatment groups were compared in relation to the ischemia related vessel (LAD vs RCX or RCA).

Results of treatment	APSAC		vs	PLACEBO		
Angiography	I		II	I		II
TWMS, LAD	10.2	*	7.6	8.2		8.4
RCX/RCA	8.5	*	6.6	7.5	#	6.5
CDS , LAD	64	*	52	64		60
RCX/RCA	79	#	67	71		69

(TWMS and CDS expressed in mean value, * p≤0.002, # p≤0.01)
Conclusions: RWM improves significantly in T treated pts, especially in LAD, which might by explained by a significant reduction in CDS. Maybe T is only indicated in treating impending anterior wall infarction.

POSTISCHEMIC ELECTRICAL STUNNING ACCOMPANIED BY STUNNED MYOCARDIUM IN ACUTE MYOCARDIAL INFARCTION

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To determine the relationship between postischemic- electrical stunning and stunned myocardium following successfull intracoronary thrombolysis (CT), 12-lead ECGs and the left ventricular function were evaluated in 95 patients (pts), evolving acute anteroseptal myocardial infarction (MI) and without prior MI. CT was performed 2 to 6 hours after onset (average: 4.1±1.5 hours). A 12-lead ECG was taken on admission, after CT, every 4 hours for 2 days, twice a day for next 3 days, and daily thereafter. Anterior regional ejection fraction (REF, by Gelberg et al) was determined from contrast left ventriculogram in 30° right anterior obilque projection 30 min after CT (REF1) and 4 weeks later (REF2) to assess regional left ventricular fucntion. All pts showed anteroapical dyskinesis after CT 30 min, suggesting postischemic myocardial stunning, and developed Q-waves in precordial leads (V1-V4). Such loss of R-wave was followed by early partial regrowth of R-wave (R-wave of >.1mV in at least 2 precordial leads) within 24 hrs in 33 pts (Group A) and by later partial R-wave regrowth in a week in 30 pts (Group B), suggesting the recovery process of postischemic electrical stunning. Seventeen pts with sustaiend Q wave despite successful CT (Group C) and 15 pts with failed CT and Q-wave MI (Group D) were also studied. Results(mean±SD):

Group	A	B	C	D
REF1(%)	8.6±10.2	9.8±11.2	5.7±8.9	3.8±8.9
REF2(%)	47.1±16.2*†	20.0±11.5#†	18.1±10.9	5.6±4.0

(*: p<.05 vs Group B, C, & D; #: p<.05 vs Group D; †: p<.05 vs REF1)
These results suggest that postischemic electrical stunning is accompanied by myocardial stunning and the early recovery of electrical stunning could be attributed to the recovery of stunned myocardium.